

Submitted:
28.05.2017
Accepted:
18.06.2017
Published:
29.09.2017

Imaging of juvenile spondyloarthritis. Part II: Ultrasonography and magnetic resonance imaging

Iwona Sudoł-Szopińska^{1,2}, Michał Znajdek¹, Piotr Gietka³,
Violeta Vasilevska-Nikodinovska^{4,5}, Lukas Patrovic⁶, Vladka Salapura⁷

¹ Department of Radiology, National Institute of Geriatrics, Rheumatology and Rehabilitation, Warsaw, Poland

² Department of Medical Imaging, Second Faculty of Medicine, Medical University of Warsaw, Warsaw, Poland

³ Clinic of Pediatrics, National Institute of Geriatrics, Rheumatology and Rehabilitation, Warsaw, Poland

⁴ University „Ss. Cyril and Methodius”, Skopje, Macedonia

⁵ University Surgical Clinic „St. Naum Ohridski” Skopje, Macedonia

⁶ MRI Department, Jessenius, Diagnostic Center, Špitálska 6, Nitra, Slovakia

⁷ University Medical Centre Ljubljana, Slovenia

Correspondence: Professor Iwona Sudoł-Szopińska, Department of Radiology, National Institute of Geriatrics, Rheumatology and Rehabilitation, Warsaw, Spartańska 1, Warszawa 02-637; e-mail: sudolszopinska@gmail.com

DOI: 10.15557/JoU.2017.0026

Keywords

juvenile
spondyloarthropathies,
ultrasonography,
magnetic resonance
imaging

Abstract

Juvenile spondyloarthropathies are mainly manifested by symptoms of peripheral arthritis and enthesitis. Early involvement of sacroiliac joints and spine is exceptionally rare in children; this usually happens in adulthood. Conventional radiographs visualize late inflammatory lesions. Early diagnosis is possible with the use of ultrasonography and magnetic resonance imaging. The first part of the article presented classifications and radiographic presentation of juvenile spondyloarthropathies. This part discusses changes seen on ultrasonography and magnetic resonance imaging. In patients with juvenile spondyloarthropathies, these examinations are conducted to diagnose inflammatory lesions in peripheral joints, tendon sheaths, tendons and bursae. Moreover, magnetic resonance also shows subchondral bone marrow edema, which is considered an early sign of inflammation. Ultrasonography and magnetic resonance imaging do not show specific lesions for any rheumatic disease. Nevertheless, they are conducted for early diagnosis, treatment monitoring and identifying complications. This article presents a spectrum of inflammatory changes and discusses the diagnostic value of ultrasonography and magnetic resonance imaging.

Ultrasonography

The range of features visible on ultrasonography is not different from those seen in adult patients with rheumatic diseases⁽¹⁾. A US examination is conducted for initial diagnosis and monitoring of treatment efficacy.

Peripheral joints

The first sign of peripheral arthritis, tenosynovitis and bursitis is the thickening of the synovial membrane resulting from synoviocyte hyperplasia and edema of the synovial subintima. It is followed by its increased vascularization and effusion that accompanies synovitis (Fig. 1). At this stage, ultrasonography enables assessment of the spectrum of inflammatory changes, their location and

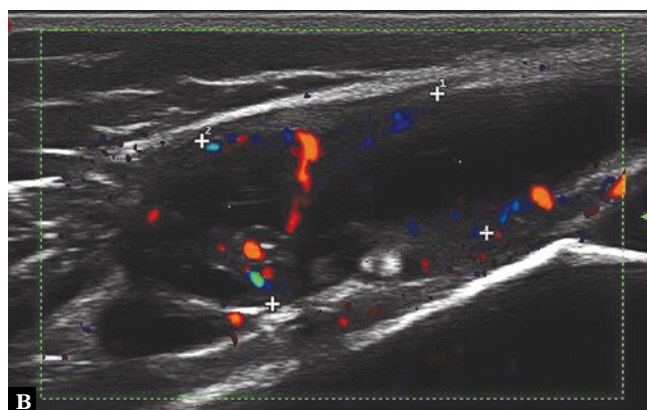
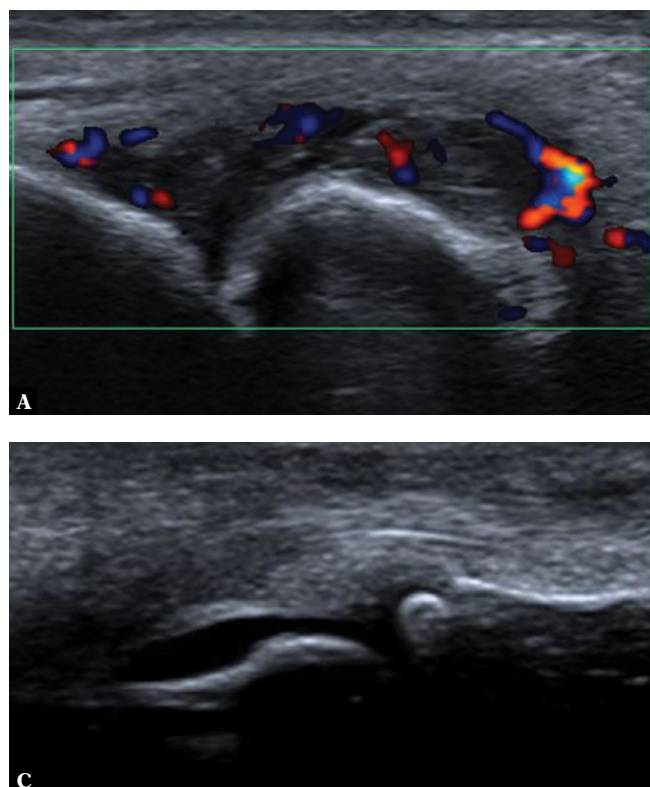
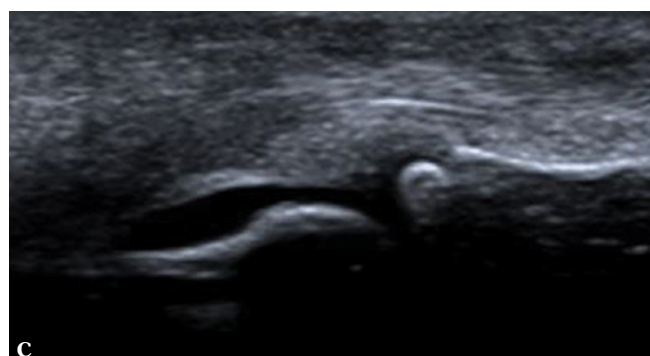


Fig. 1. Effusion, thickening and enhanced blood flow in the synovium of: **A.** 2th MCP joint in the right hand of a 17-year-old girl with JSpA; **B.** knee joint in a 10-year-old girl with JSpA; **C.** effusions, no synovial pathology in the MCP 3 joint of the left hand in a 11-year-old girl



advancement. Moreover, based on a US examination, a child can be selected for a decompression procedure or deemed ineligible if US shows bands of fibrosis, thickened synovium or that the joint cavity is filled with multiple textured elements.

As the disease develops, erosions form. Initially, they can be seen at the border of the articular surface covered with cartilage and the site of joint capsule attachment (so-called *bare area*). They are called marginal erosions (Fig. 2, 3). When the articular cartilage is destroyed, subchondral erosions appear. By contrast with radiography, ultrasonography can identify the first stages of articular cartilage destruction, in the form of its increased echogenicity, as well as deeper and deeper defects caused by pannus invasion.

Inflammatory cysts (also called geodes) reflect the presence of inflammatory infiltrates in the subchondral bone tissue (Fig. 2).

In chronic conditions, the synovial membrane becomes hypertrophic until it assumes forms of various shapes; in this case, so-called rice bodies develop as a result of fragmentation of the hypertrophic synovium. Enhanced synovial vascularization reduces or subsides completely in the case of a positive response to treatment. Persisting intensive synovial vascularization can be an indication for surgical or radioisotope synovectomy, the latter should be conducted under US guidance⁽²⁾.

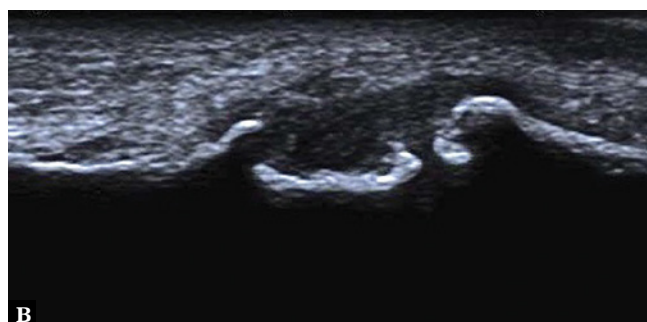


Fig. 2. US: **A.** small marginal erosion and large geode in the head of the 5th metacarpal bone in the right hand of a 16-year-old boy; **B.** large erosion in the 2nd MTP joint in the right foot of a 14-year-old-boy with JSpA

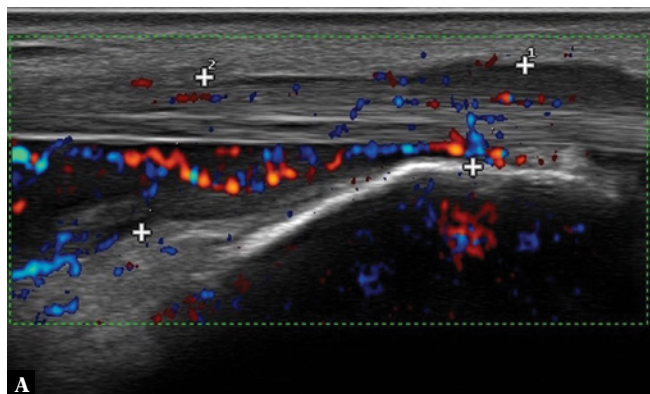


Fig. 3. Thickened and intensively vascularized synovium of the tibialis posterior tendon sheath in a 16-year-old girl with JSpA

Tendon sheaths

Tenosynovitis (tenovaginitis) is manifested by thickened synovium and its increased vascularization, usually with accompanying effusion (Fig. 3). Tendinitis (also tendonitis) is a complication of tenosynovitis. A tendon that is weakened by inflammation may undergo damage, including complete tear.

Bursae

In the case of bursitis, the following signs can be observed: synovial thickening, hypervascularization and effusion (Fig. 4). A bursa that is filled with fluid and hypertrophic synovium can rupture (usually Baker's cyst). Persisting bursitis (of e.g. Achilles tendon or deep infrapatellar bursa) may involve the adjacent tendon, leading to its damage and causing erosions in the bony wall of the bursa. The administration of a steroid drug into an inflamed bursa complicated with tendinitis may lead to tendon damage.

Intraarticular and extraarticular fat tissue

It has been demonstrated in adults with rheumatoid arthritis that fat tissue is another site (next to the synovium and subchondral bone tissue) for inflammatory infiltrates, the

cells of which participate in the joint destruction process⁽³⁾. No such studies have been conducted for juvenile spondyloarthropathies or juvenile idiopathic arthritis. Nevertheless, a US scan shows edema and hypervascularization of the intra- and extraarticular fat tissue even more frequently in children than in adults (author's own unpublished observations), which suggests that identical pathological processes are involved⁽⁴⁾ (Fig. 5).

Enthesopathies

In a US examination, pathological entheses are thickened (edema) and hypoechoic. Moreover, one can observe delaminations, areas of degeneration and vessels of the inflammatory-repair process. The bone layer may present erosions and cysts. In adult patients, pathological lesions in entheses cannot be discriminated from far more common microinjuries or degenerative changes, frequently seen also in healthy individuals, since they produce identical US images. In children, however, the probability of injuries or entheses degeneration is low and therefore an abnormal image, particularly hypervascularization, in combination with other clinical data can be interpreted as enthesitis (Fig. 6). This issue requires further studies particularly because SEA or ERA are, according to clinical data, crucial JSpA entities (see part 1 of the publication).

Magnetic resonance imaging

In addition to identical to US ability to visualize features of synovitis, tenosynovitis, bursitis, enthesopathies and fat tissue pathologies, magnetic resonance imaging (MRI) enables assessment of^(5,6) (Fig. 7):

- bone marrow edema, which is a pre-erosional condition and a sign of inflammation;
- articular cartilage in its entire range;
- changes in the spine and spinal cord;
- activity of the involved synovium and subchondral bone tissue in a contrast-enhanced examination.

MRI is more sensitive in assessment of inflammatory and destructive changes in JIA and JSpA than physical examination, radiographs or US. MRI protocols enable

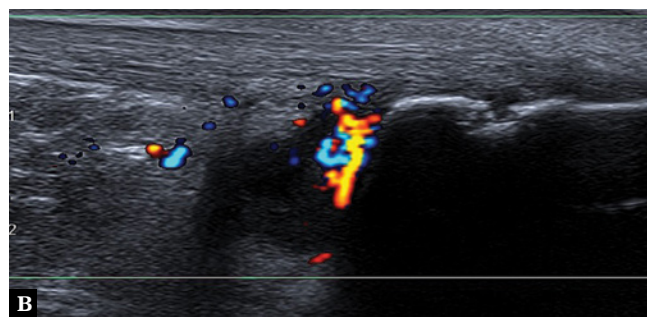
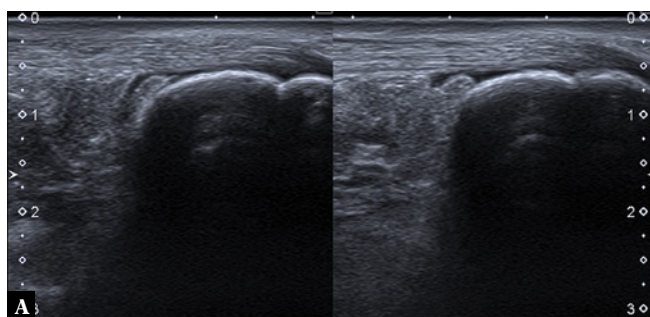


Fig. 4. Bursitis of the left Achilles tendon: **A.** Bilateral slight effusion in the Achilles tendon bursae in a 13-year-old girl, no synovial pathology, rounded fat fold of the left bursa – chronic inflammatory changes; **B.** thickened and intensively vascularized synovium of the Achilles tendon bursa in a 15-year-old girl, erosion in the bony wall of the bursa

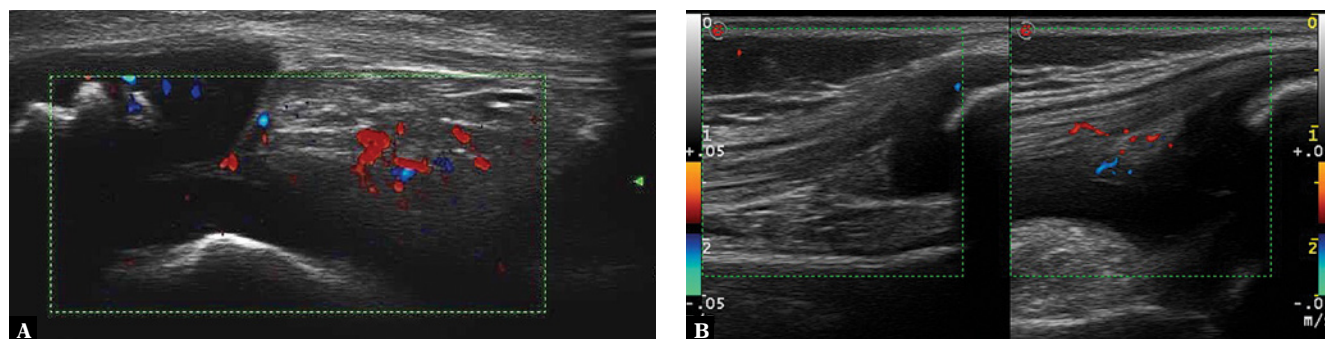


Fig. 5. Edema, features of enhanced vascularization of the intraarticular fat: A. Hoffa's fat pad in a 10-year-old girl with JSpA; B. fat tissue of the left quadriceps femoris tendon / suprapatellar fat pad (normal in the right joint)

examinations without the need of administering an anesthetic and sedative agents even in young children^(7–12). However, the available literature contains few papers devoted to MRI in children with JSpA. Bollow et al.⁽¹⁰⁾ scanned sacroiliac joints (SIJs) of 100 children with suspected JSpA and in 30 controls. Active inflammatory changes were detected in 42.9% of children with JSpA while radiographs were normal. In the entire group of 130 children, active and chronic inflammatory lesions were detected in 41% of children whereas chronic changes were visualized only in patients with clinically suspected JSpA. Chronic inflammatory changes were detected on MRI more frequently than on radiographs (14.3% vs 6.7%), which is consistent with our studies conducted in adults⁽¹¹⁾. Children with suspected JSpA, in whom active lesions were detected in MRI, also had considerably higher levels of C-reactive protein ($p=0.01$) and significantly longer medical history ($p=0.01$) compared to children without features of sacroiliitis in MRI: 63.2 ± 44.1 months vs 28.2 ± 29.7 months. The onset of symptoms in the first group took place at the age of 8.5 ± 3 years, and in the group of children without sacroiliitis in MRI at the age of 11.0 ± 3.2 years⁽¹⁰⁾.

Another publication⁽¹²⁾ demonstrated higher sensitivity of MRI compared with radiography in diagnosing sacroiliitis. Apart from the features of sacroiliitis, MRI also showed signs of enthesitis in the pelvis (pubic symphysis 91%, greater or lesser trochanter 55%, hip joint 45%, iliac crest 27%, ischial tuberosity 27%).

Herregods et al.⁽¹³⁾ conducted MRI examinations of the sacroiliac joints in 80 children with clinically suspected JSpAs. Sacroiliitis was not detected in most patients. Bone marrow edema (BME) was found in 16 of 80 children (20%), high signal in the articular cavity in 18 of 80 children (22.5%) and sacroiliac capsulitis in 6 children (7.5%). The authors found that MRI performed before contrast enhancement was consistent with contrast-enhanced examination in detecting BME and capsulitis. A high signal in the sacroiliac joint cavity was present in 22.5% of patients, including contrast enhancement in 83%. The authors did not find any significant benefits of contrast-enhanced MRI and claimed that, as in adults, STIR/TIRM (short tau inversion recovery/turbo inversion recovery magnitude) se-

quences were sufficient to make a diagnosis. JSpA was not ultimately confirmed in patients with a high signal in the joint cavity with no other features of sacroiliitis. Different conclusions were drawn by Lin et al.⁽¹⁴⁾ They stated that, by contrast with adults, capsulitis in children can be an independent factor of inflammation, without accompanying BME.

Rachlis et al.⁽¹⁵⁾ demonstrated that whole body MRI is superior to pelvic MRI in assessing inflammatory and enthesopathic changes in the hip, sacroiliac and spinal joints. The authors did not confirm the features of enthesitis suspected in a clinical examination.

Conclusion

Plain radiography, with its role to rule out malignancies, trauma or specific inflammations, still remains a standard in the diagnostic process of early inflammatory changes in the course of JSpA. In early stages of JSpA, radiography is usually negative or reveals features of osteoporosis, increased density and extended soft tissue shadow, followed by erosions and cysts. Typically, lower extremity joints are involved (see part 1 of the article). The next ex-

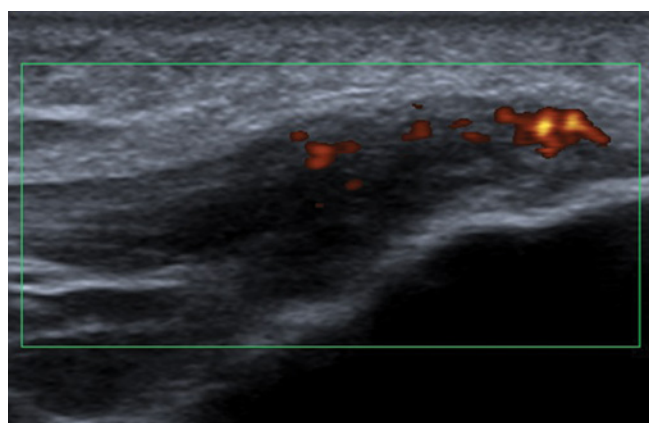


Fig. 6. Enthesitis-like changes of the tibial enthesis of the patellar tendon in a 15-year-old HLA-B27+ boy: swollen enthesis with lower echogenicity and hypervascularization

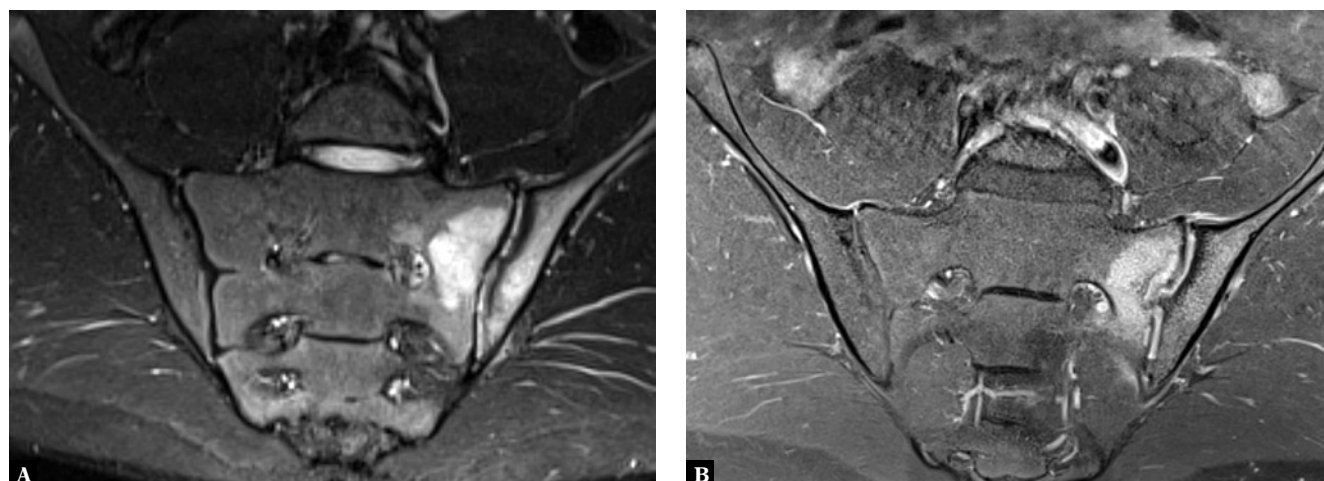


Fig. 7. MRI of the sacroiliac joints in a 12-year-old boy with suspected sacroiliitis. Coronal oblique planes. **A.** T2-weighted TIRM, **B.** T1-weighted FS CM (contrast medium): bilateral bone marrow edema, more marked in the left joint, thickened contrast-enhancing synovium

amination is usually ultrasonography. Its aim is to show early inflammatory lesions in peripheral joints, tendon sheaths, bursae and entheses. Additionally, MRI presents bone marrow edema in the sacroiliac joints in the course of sacroiliitis and as well as atlanto-occipital pathologies. Bollow *et al.*⁽¹⁰⁾ compared a population of HLA-B27-positive children and adults with peripheral arthritis in terms of the co-occurrence of sacroiliitis. It was detected in 70–80% of adults, and in merely 35% of children⁽¹⁰⁾. Despite the fact that the SIJs are rarely involved in early JSpAs, children and adolescents with a suspected disease should be referred to an MRI examination for early identification of inflammatory changes and swift implementation of treatment that will prevent the progression of inflammation in the axial spine. It is possible that the detection of the signs of sacroiliitis in MRI at an early stage of the disease is another unfavorable prognostic factor indicat-

ing a severe course of the disease and possible transformation into AS in adulthood⁽¹⁰⁾.

There is a need not only for further research on this issue, but also for an update of JSpA diagnostic algorithms. Another suggestion of JSpA clinical criteria⁽¹⁶⁾ is a step towards this goal since they include MRI is the diagnostic process of sacroiliitis. Jaremko *et al.*⁽¹⁷⁾ compared radiographs and MRI of the sacroiliac joints and demonstrated superiority of MRI.

Conflict of interest

Authors do not report any financial or personal connections with other persons or organizations, which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

References

1. Sudół-Szopińska I, Jans L, Teh J: Rheumatoid arthritis: what do MRI and ultrasound show. *J Ultrason* 2017; 17: 5–16.
2. Ćwikła JB, Żbikowski B, Kwiatkowska B, Buscombe JR, Sudół-Szopińska I: Radiosynovectomy in rheumatic diseases. *J Ultrason* 2014; 14: 241–251.
3. Sudół-Szopińska I, Kontny E, Zaniewicz-Kaniewska K, Prohorec-Sobieszek M, Saied F, Maśliński W: Role of inflammatory factors and adipose tissue in pathogenesis of rheumatoid arthritis and osteoarthritis. Part I: Rheumatoid adipose tissue. *J Ultrason* 2013; 13: 192–201.
4. Sudół-Szopińska I, Hrycaj P, Prohorec-Sobieszek M: Role of inflammatory factors and adipose tissue in pathogenesis of rheumatoid arthritis and osteoarthritis. Part II: Inflammatory background of osteoarthritis. *J Ultrason* 2013; 13: 319–328.
5. Mandl P, Navarro-Compán V, Terslev L, Aegerter P, van der Heijde D, D'Agostino MA *et al.*: EULAR recommendations for the use of imaging in the diagnosis and management of spondyloarthritis in clinical practice. *Ann Rheum Dis* 2015; 74: 1327–1339.
6. Sudół-Szopińska I, Jurik AG, Eshed I, Lennart J, Grainger A, Østergaard M *et al.*: Recommendations of the ESSR arthritis subcommittee for the use of magnetic resonance imaging in musculoskeletal rheumatic diseases. *Semin Musculoskelet Radiol* 2015; 19: 396–411.
7. Colebatch-Bourn AN, Edwards CJ, Collado P, D'Agostino MA, Hemke R, Jousse-Joulin S *et al.*: EULAR-PRES points to consider for the use of imaging in the diagnosis and management of juvenile idiopathic arthritis in clinical practice. *Ann Rheum Dis* 2015; 74: 1946–1957.
8. Hemke R, Kuijpers TW, Nusman CM, Schonenberg-Meinema D, van Rossum MA, Dolman KM *et al.*: Contrast-enhanced MRI features in the early diagnosis of Juvenile Idiopathic Arthritis. *Eur Radiol* 2015; 25: 3222–3229.
9. Nusman CM, Ording Muller LS, Hemke R, Doria AS, Avenarius D, Tzaribachev N *et al.*: Current status of efforts on standardizing magnetic resonance imaging of juvenile idiopathic arthritis: Report from the OMERACT MRI in JIA Working Group and Health-e-Child. *J Rheumatol* 2016; 43: 239–244.
10. Bollow M, Biedermann T, Kannenberg J, Paris S, Schauer-Petrowski C, Minden K *et al.*: Use of dynamic magnetic resonance imaging to detect sacroiliitis in HLA-B27 positive and negative children with juvenile arthritides. *J Rheumatol* 1998; 25: 556–564.
11. Sudół-Szopińska I, Kwiatkowska B, Włodkowska-Korytkowska M, Matuszewska G, Grochowska E: Diagnostics of sacroiliitis accor-

- ding to ASAS criteria: A comparative evaluation of conventional radiographs and MRI in patients with a clinical suspicion of spondyloarthropathy. Preliminary results. *Pol J Radiol* 2015; 80: 266–276.
12. Tse SML, Laxer RM: New advances in juvenile spondyloarthritis. *Nat Rev Rheumatol* 2012; 8: 269–279.
13. Herregods N, Jaremko JL, Baraliakos X, Dehoorne J, Leus A, Verstraete K *et al.*: Limited role of gadolinium to detect active sacroiliitis on MRI in juvenile spondyloarthritis. *Skeletal Radiol* 2015; 44: 1637–1646.
14. Lin C, MacKenzie JD, Courtier JL, Gu JT, Milojevic D: Magnetic resonance imaging findings in juvenile spondyloarthropathy and effects of treatment observed on subsequent imaging. *Pediatr Rheumatol Online J* 2014; 12: 25.
15. Rachlis AC, Babyn PS, Lobo-Mueller E, Benseler SM, Stimec J, Anderson M: Whole body MR imaging in juvenile spondyloarthritis: Will it provide vital information compared to clinical exam alone. *Arthritis Rheum* 2011; 63: S292.
16. Sezen M, Barut K, Açikel C, Kasapcopur O: The new proposal classification criteria for juvenile spondyloarthropathies. *Pediatr Rheumatol Online J* 2014; 12 (Suppl. 1): P45.
17. Jaremko JL, Liu L, Winn NJ, Ellsworth JE, Lambert RG: Diagnostic utility of magnetic resonance imaging and radiography in juvenile spondyloarthritis: evaluation of the sacroiliac joints in controls and affected subjects. *J Rheumatol* 2014; 41: 963–970.